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EFFECT OF EARLY FEED RESTRICTION IN MALE BROILER CHICKS ON PLASMA METABOLIC HORMONES DURING FEED RESTRICTION AND ACCELERATED GROWTH

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Abstract—1. Plasma GH was greater ($P < 0.05$) on day 12 in *ad libitum*-fed birds compared to restricted chicks. Conversely, maximum GH levels were found to occur in the nutrient restricted chicks during the period of accelerated growth (day 42).

2. A significant decline in circulating insulin concentrations with advancing age was evident in both *ad libitum*-fed and restricted chicks.

3. Feed restriction significantly suppressed circulating T3 in restricted chicks, with concentrations returning to control levels upon refeeding.

4. A significant increase in T4 with advancing age was evident in both treatment groups, with T4 being significantly greater in controls compared to restricted chicks at 54 days of age.

INTRODUCTION

Recent studies in this laboratory (Plavnik *et al.*, 1986) and elsewhere (Plavnik and Hurwitz, 1985) demonstrated that early feed restriction of broiler chicks will change nutrient partitioning during the later stages of growth. Although feed restriction initially reduces growth, chicks grow more rapidly than controls during refeeding. This rapid growth is termed catch-up or compensatory growth. This phenomenon has been observed in other species as well (Wilson and Osbourn, 1960). Limiting nutrient intake may also increase longevity (Yu *et al.*, 1982). To account for the phenomenon of accelerated growth, major changes in the composition of body weight gain, increased efficiency of energy utilization, a reduction in maintenance energy requirements, an enhancement in food utilization or a combination of these adaptations may have taken place. To gain insight into the nature of these metabolic alterations, selected hormones known to be important in the regulation of growth and metabolism in birds were monitored.

MATERIALS AND METHODS

Sixty 1-day-old Ross broiler male chicks were raised in battery-brooders and fed a 21% protein starter diet containing 2.95 kcal/g metabolizable energy until 6 days of age (Plavnik *et al.*, 1986). On day 6, chicks were divided into two treatments according to individual body weights: (1) control (CTL), which had access to feed at all times, (2) R6, which was restricted in feed intake from days 6–12 of age. Feed restriction and subsequent refeeding were conducted as previously described (Plavnik *et al.*, 1986). On day 6 post-hatching, R6 chicks were fed daily for 6 days an amount of feed that only met the maintenance energy requirement at 6 days of age. At 4 weeks of age, chicks were transfered to grower batteries and fed an 18% protein grower diet. Chicks were raised under constant light, constant temperature (24° C), and with free access to water. Body weights and feed intakes were recorded weekly. The effects of restriction treatment on rate and efficiency of gain and on composition of gain have been published previously (Plavnik *et al.*, 1986).

Heparinized blood samples were drawn via cardiac puncture on day 5 (to determine pretreatment hormone levels) and at 12 days (at the conclusion of the restriction period) and at 18 days of age. Samplings at 42 and 54 days of age were drawn from the brachial vein. All blood samplings were conducted in less than 1 min after removing the chick from its pen. Samplings were carried out between 0900 and 0930 hrs, which was *ca.* 120 min after the birds were fed. Plasma was harvested and kept frozen at −30°C until analyzed.

Hormone analyses on the plasma samples were performed by radioimmunoassay (RIA) as duplicate determinations. For a particular hormone, samples were analyzed in one assay to avoid interassay variation. Insulin was determined by an homologous RIA for chicken insulin (McMurtry *et al.*, 1983). The sensitivity of this assay is 10 pg/tube. The glucagon RIA was conducted as previously described (Allen and McMurtry, 1984). Porcine glucagon 125-I was purchased from Cambridge Medical Diagnostics (Billerica, MA). The primary antisera (pancreatic-specific) and porcine glucagon standard were obtained from Novo Research, Copenhagen, Denmark. The sensitivity of the glucagon RIA was 9.7 pg/tube. Growth hormone was determined utilizing recombinant-derived chicken hormone (Proudman, 1984). Plasma triiodothyronine (T3) and thyroxine (T4) were determined as described by May (1978). L-T3 and L-T4 for standards were obtained from Sigma Chemical Co., St Louis, MO and prepared in charcoal-stripped chicken plasma. Radiolabelled T3 and T4 were purchased from Cambridge Medical Diagnostics and the primary antisera from Cooper Biochemical, Malvern, PA.

Statistical analyses were performed to determine treatment effects utilizing the least-square routine in the General Linear Model (SAS, 1979).

RESULTS

There was a significant ($P < 0.05$) decline in insulin levels as the birds matured. Circulating insulin levels were greatest on day 5 of age and least on day 42 in

both treatment groups (Table 1). There was no difference between the *ad libitum*-fed CTL and restricted birds at any age.

Plasma glucagon concentrations were significantly ($P < 0.05$) lower in R6 chicks on day 12 (day 6 of feed restriction) and day 42, compared to *ad libitum*-fed CTL (Table 1).

In contrast, a significant change over time was noted in circulating T4 levels (Table 1). An increase in T4 occurred in both groups as the birds matured, with levels being the lowest on day 12, and gradually increasing thereafter. There was a significant difference ($P < 0.05$) in T4 between CTL and restricted chicks at 54 days of age.

Feed restriction decreased T3 ($P < 0.05$; Table 1). On day 12 (6 days of restriction), T3 concentrations averaged 240 pg/ml in restricted chicks compared to 380 pg/ml in control chicks. Plasma T3 increased in R6 birds following the return to *ad libitum* feeding, as no difference in T3 concentrations was observed on day 18 between the groups. A significant ($P < 0.05$) decline in T3 was noted over time in both groups (day 54).

Maximum circulating GH (24.2 ng/ml) was noted in the CTL chicks on day 12, whereas this pattern was delayed until 42 days of age in the R6 birds. A significant ($P < 0.05$) age–GH interaction was noted in both treatment groups, as levels declined with advancing age.

Table 1. The effect of early in life nutrient restriction on metabolic hormone concentrations in broiler chicks

Hormone	d5		d12		d18		d42		d54	
	CTL	R6	CTL	R6	CTL	R6	CTL	R6	CTL	R6
Insulin (ng/ml)	1.86 ± 0.29^{a}	1.77 ± 0.18^{a}	$1.35 \pm 0.20^{b,c}$	$1.37 \pm 15^{b,c}$	1.15 ± 0.11^{c}	$1.39 \pm 0.26^{a,b,c}$	0.48 ± 0.10^{d}	0.39 ± 0.09^{d}	1.11 ± 0.14^{c}	0.95 ± 0.20^{c}
Glucagon (pg/ml)	205 ± 15^{a}	174 ± 25^{a}	199 ± 12^{a}	175 ± 9^{b}	173 ± 16^{a}	187 ± 5^{a}	222 ± 24^{a}	182 ± 7^{b}	161 ± 9^{b}	$169 \pm 11^{a,b}$
Thyroxine (ng/ml)	8.7 ± 0.9^{a}	8.9 ± 0.4^{a}	6.1 ± 1.3^{a}	6.9 ± 0.9^{a}	7.1 ± 0.5^{a}	8.5 ± 1.1^{a}	11.8 ± 0.4^{b}	12.2 ± 0.5^{b}	18.6 ± 1.7^{c}	12.2 ± 0.7^{b}
Triiodothyronine (pg/ml)	425 ± 52^{a}	434 ± 61^{a}	380 ± 37^{a}	240 ± 12^{b}	284 ± 26^{b}	$311 \pm 87^{a,b}$	237 ± 15^{c}	234 ± 19^{c}	$217 \pm 11^{c,d}$	$193 \pm 17^{c,d}$
Growth hormone (ng/ml)	13.3 ± 3.2^{a}	8.7 ± 3.6^{a}	24.2 ± 4.9^{b}	12.2 ± 2.4^{a}	14.8 ± 2.1^{a}	16.1 ± 3.7^{a}	9.5 ± 3.3^{a}	26.7 ± 4.9^{b}	3.5 ± 0.4^{c}	4.9 ± 1.6^{c}

^{a,b,c,d}Means ± SEM of 6 observations. Means within a row with the same superscript are not significantly different ($P < 0.05$).
CTL = control-ad libitum-fed chicks; R6 = feed restricted chicks; restriction conducted as described in Materials and Methods.

DISCUSSION

The mechanisms regulating both lean and adipose tissue development are not as well understood in birds as in mammals. The lack of information may be mainly due to a scarcity of data concerning metabolic regulators. As previously reported, when feed intake is limited in young chicks, the birds exhibit catch-up or accelerated growth at 3–4 weeks of age. At maturity, the restricted birds are equivalent in body weight but have smaller abdominal fat pads and total carcass fat (Plavnik *et al.*, 1986). Clearly, significant changes in the partitioning of nutrients to favor lean tissue accretion at the expense of metabolic energy to support fat tissue synthesis has occurred. The present study was conducted to ascertain if changes in endocrine function were associated with the metabolic and tissue changes that occur in restricted chicks. Modulation of tissue sensitivity as measured by target tissue interactions (receptor characterization) was not considered in this study. Post-receptor adaptations are considered in a companion report detailing metabolism of selected tissue from feed restricted broilers (Rosebrough *et al.*, 1986).

In recent years, considerable research has centered on ways of regulating adipose tissue development in broilers either through the use of nutritional control mechanisms or via schemes of genetic selection (Simon and Brisson, 1972; Leclercq, 1984). Most of these studies produced questionable results because the birds responded by either failing to grow at the normal rate or becoming obese. Plavnik *et al.* (1986) demonstrated that many of the drawbacks were overcome if feed restriction was conducted at a specific time and of short duration early in life.

Numerous studies have shown that changes occur in thyroid function when chicks are subjected to periods of fasting and refeeding (Harvey and Klandorf, 1983; Alster and Carew, 1984; Decuypere and Kuhn, 1984). During fasting, circulating T3 is depressed while T4 is increased. A similar response was observed in the restricted chicks in that T3 was suppressed during restriction but returned to normal levels following realimentation. Feed restriction is not fasting, suggesting that the hypothalamo-pituitary-thyroid axis in birds is extremely sensitive to the slightest deprivation in food. A reduction in monodeiodinase activity during restriction may account for part of the change in T3 (Decuypere and Kuhn, 1984). Similarly, insulin concentrations are known to be suppressed following fasting, and rebound to supranormal levels following refeeding (Rosebrough *et al.*, 1984). Conversely, glucagon secretion is enhanced during fasting and depressed following refeeding (Hazelwood, 1980). Similar changes in the two pancreatic hormones were not observed in this study, suggesting that feed restriction in the present study was not severe enough to elicit such responses. It is interesting to note that insulin levels tended to be elevated early in life, and then declined as the birds grew older. A similar decline with age in circulating insulin concentrations has been recently reported for the domestic fowl (Vasilatos-Younken, 1986). As suggested by the same author, a decrease in blood insulin with age is apparently linked to a concurrent decline in feed intake.

GH is elevated in growing turkeys during a long-term feed restriction (Proudman and Opel, 1981). Total fasting (Harvey *et al.*, 1978) and intermittent feeding regimes in chickens (Nir *et al.*, 1983) also increase plasma GH concentrations. It is possible that a major reason for the results of the present study may reside in the method of feeding practised. Limiting feed intake for a short duration does not appear to place stress on the endocrine system that total feed restriction does. Compared to the restriction regimen utilized in the present study in which body weight was maintained, total nutritional abstinence may be the stimulus necessary to induce significant endocrine adaptations.

As noted by Scanes and Harvey (1981) and in the present study, plasma GH levels were high during the period of neonatal growth in the *ad libitum*-fed birds, and then declined as the birds matured. It is interesting to note that early feed restriction delayed the normal elevation in GH concentrations until the chicks were in a period of most rapid growth at 3 and 4 weeks of age (Plavnik *et al.*, 1984). The poor relationship between GH levels and growth (Burke and Marks, 1982) makes causal relationship doubtful in chicks. More frequent sampling during this period may be informative as to whether any cause-effect relationship exists between GH and growth. As suggested by King and May (1984) and supported by this study, thyroid status may be a better index of growth rate in birds than GH.

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